OBSERVATIONS ON THE CLINICAL USE OF PAIRED ELECTRICAL STIMULATION OF THE HEART*

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INDICATIONS for the clinical use of the method of paired or coupled electrical stimulation of the heart[1-5] are: 1) the need for slowing of an uncontrollable tachycardia, regardless of its origin; 2) the need for increase of the force of ventricular contraction, or 3) the combination of the first and second. The purpose of this report is to point out two special clinical applications of paired stimulation: one pertaining to atrial, the other to ventricular pacing.

1) *A bedside method of coupled atrial stimulation for the purpose of slowing the ventricular rate during uncontrollable sinus tachycardia.* Pursuing the experimental studies reported from this laboratory[1] we have been trying to develop a simple bedside technique of paired atrial stimulation that does not require fluoroscopic control for the introduction of the electrode catheter and would permit slowing of the ventricular rate in marked sinus tachycardia. Recording of the intracavitary electrocardiogram as the catheter is advanced into the right atrium permits localization and proper placement of the catheter electrode. The paired atrial stimulus must occur late enough to fall outside of the refractory period of the atrial muscle and early enough to find the A-V junction still refractory in order to prevent its propagation to the ventricles. This can be accomplished by paired atrial stimulation with the dominant atrial rate slightly exceeding that of the sinus node[1] or, to achieve maximal slowing with a given sinus rate, by coupling the nonconducted premature atrial stimulus to the conducted sinus beat, using the R wave of the latter to trigger the premature atrial response.† During such nonconducted atrial bigeminy (Figures 1*a* and 1*b*) the ventricular cycle is lengthened by a time interval equal to the sum of the "coupling" of the premature atrial beat, the conduction time of the premature impulse to

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† Following a suggestion of Dr. E. M. McNally.

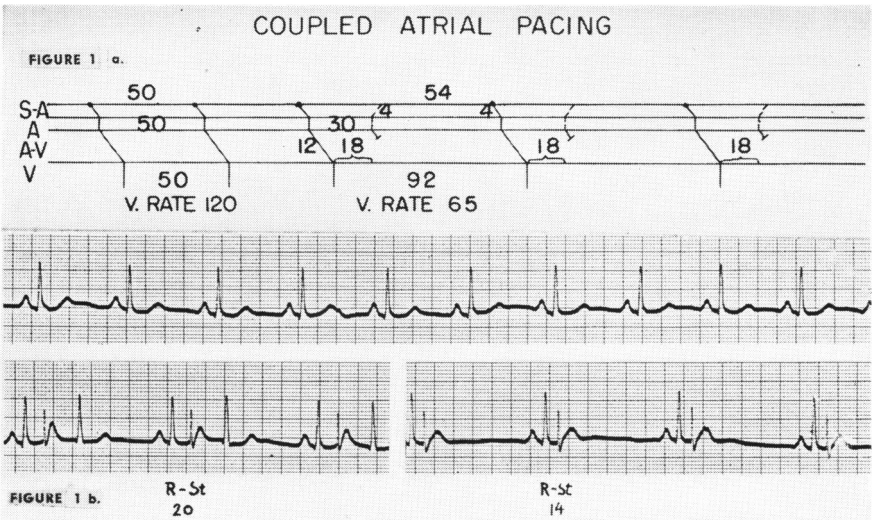

Fig. 1*a*. Mechanism of ventricular slowing by coupled atrial pacing. The conventions in the diagram are as follows: *S-A* represents the spread of the impulse from the sinus node to the atria (*A*) and vice versa, *A-V* from the atria to the ventricles (*V*). Intervals in hundredths of a second. Beginning with the 3d ventricular beat, ventricular activation triggers a premature atrial beat (broken line) after a delay of 0.18 sec. The latter (*1*) discharges the sinus node and resets its timing, and (*2*) is not propagated to the ventricles because of the refractoriness of the *A-V* junction. Thus, the ventricular cycle lengthens (the ventricular rate retards).

Fig. 1*b*. Portions of lead II obtained on a 24-year-old patient with mitral stenosis. Catheter electrode in the right atrium. *Top:* control, sinus rhythm, rate 68. *Bottom:* coupled atrial pacing. With a delay (R-St) between R of the spontaneous beat and the electrical stimulus (St) set at 0.20 sec. the premature atrial impulses are conducted to the ventricles, resulting in bigeminal rhythm of both atria and ventricles (av. rate 78). With an R-St delay of 0.14 sec. the premature atrial responses are no longer propagated to the ventricles and the ventricular rate is retarded to 43.

the natural pacemaker, and the S-A conduction time of the subsequent sinus impulse plus the lengthening of the S-A cycle due to depression of the pacemaker following its premature extraneous discharge. This procedure should result in at least 30 per cent slowing of the ventricular rate.

The method of paired atrial stimulation is presumably associated with postextrasystolic potentiation of the atrial contraction. However, when ventricular bigeminy is avoided it does not provide any potentiation of ventricular systole; if ventricular slowing obtained in this manner proves insufficient in improving cardiac output the catheter can then be advanced under fluoroscopic control into the right ventricle for paired or coupled ventricular stimulation. It should be emphasized that ventricular slowing accomplished by paired atrial stimulation has

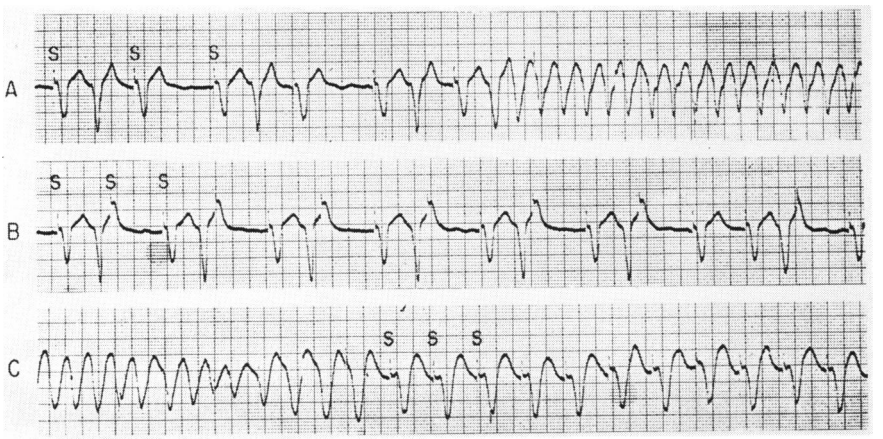

Fig. 2. Portions of lead II obtained on an 80-year-old patient with recent myocardial infarction and Stokes-Adams attacks. Atrial fibrillation with complete *A-V* dissociation. Ventricular pacing with single stimuli (*S*) via transvenous pacemaker. Spontaneous ectopic impulse formation gives rise to (interpolated) ventricular premature systoles in *A*, to intermittent bigeminy in *B* and at times initiates runs of ventricular tachycardia (end of *A* and beginning of *C*). After increasing rate of ventricular pacing to 115 (end of *C*) premature beats remained suppressed.

the advantage of avoiding the O_2 consumption of the "ineffective" ventricular response during paired ventricular stimulation.

2) *Indications for paired stimulation in patients with Stokes-Adams disease.* The use of paired ventricular stimulation in patients with advanced A-V block is exemplified by the following two cases.

Case 1. An 80-year-old female patient with recent myocardial infarction developed complete A-V dissociation with a slow ventricular rate and Stokes-Adams attacks. A transvenous electrode catheter was introduced into the right ventricle and connected to an external battery-powered pacemaker. While being paced with single stimuli the patient had frequent ventricular premature systoles, mostly in the form of bigeminy with a tendency to runs of ventricular tachycardia (Figure 2). Although acceleration of the rate of pacing from 62 to 115 abolished the spontaneous ectopic impulse formation, her blood pressure remained unobtainable in spite of the infusion of norepinephrine. An attempt with paired stimulation appeared justified in order to prevent spontaneous tachycardia and, in addition, to obtain postextrasystolic potentiation of ventricular contraction. In contrast to spontaneous bigeminy during single pacing with slow rates, induced bigeminy with a different (shorter) coupling tended to prevent a repetitive response, or ventricular

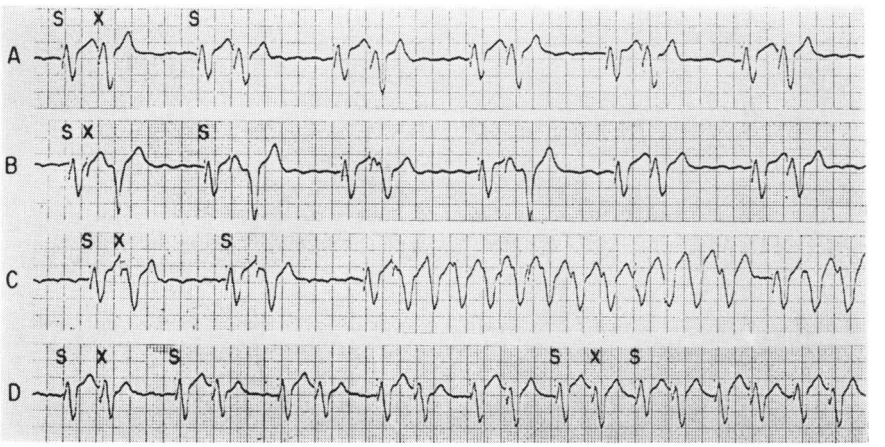

Fig. 3. Same patient as in Fig. 2. Paired ventricular stimulation (lead II). Varying driving rates (*S-S*) and varying coupling of premature stimuli (*X*). When the *S-X* interval was kept very short and *X* became ineffective (*B*) bigeminy due to spontaneous ventricular premature systoles and tendency to ventricular tachycardia (not illustrated, cf. Fig. 2) reappeared when *X* fell on top of T (*C*) it tended to initiate runs of ventricular tachycardia; when the *S-X* interval was longer but still shorter than the coupling of the spontaneous premature systoles, and at the same time the driving rate was accelerated to 62 (*D*) ventricular tachycardia was prevented.

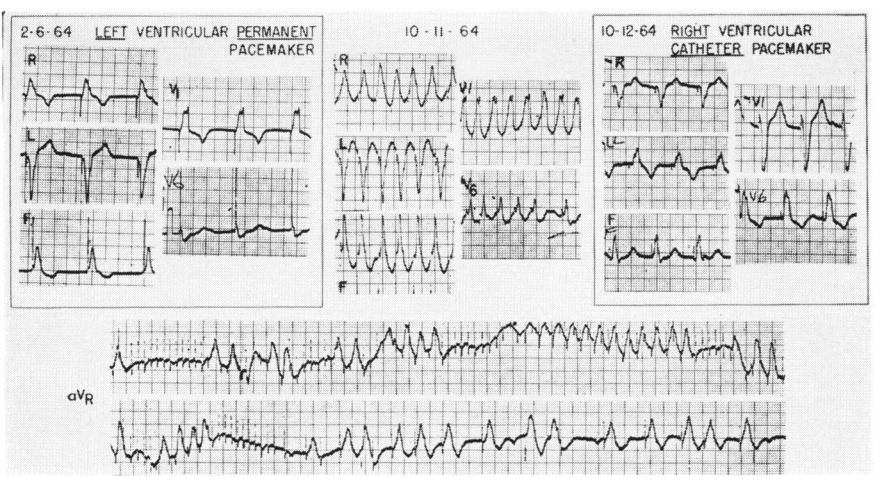

Fig. 4. Portions of records obtained on a 66-year-old patient with Stokes-Adams disease, paced artificially by an implanted left ventricular pacemaker (2-4-64) and later right ventricular pacemaker (10-12-64). "Runaway" pacemaker on 10-11-64 with intermittent rapid stimulation (rate 600) and rapid ventricular response (up to 300) presents an indication for paired ventricular stimulation via transvenous right ventricular pacemaker to slow the ventricular rate.

tachycardia (Figure 3). However, pulse and blood pressure remained unobtainable and the patient died.

Case 2. A 66-year-old patient with Stokes-Adams disease, previously reported[6] because of the unusual association of her syncopal attacks (paroxysmal ventricular fibrillation) with micturition, developed eight months after satisfactory functioning of an implanted left ventricular pacemaker (model of Chardack-Greatbatch) a rare type of pacemaker failure, the so-called "runaway pacemaker"[7] with stimulation rates from 88 to 600 and a ventricular rate up to 300 (Figure 4). Before the malfunctioning electric pacemaker was disabled the patient had to be provided with a transvenous right ventricular pacemaker. As soon as the latter was in place, paired right ventricular stimulation appeared the method of choice—if not the only method—to retard the excessive ventricular rate produced by the left ventricular pacemaker until the surgeon could disable the latter. Actually, in our patient a stimulator for paired stimulation was not immediately available; fortunately the patient survived the delay until the malfunctioning pacemaker was disabled and the transvenous pacemaker took over. It is surprising that the rapid stimulation of the ventricle did not induce ventricular fibrillation in this patient whose previous syncopal attacks had been documented as due to ventricular fibrillation. The feasibility of the therapeutic approach, namely retardation of a ventricular tachycardia produced by a left ventricular pacemaker, was shown in an experiment by Lopez *et al.*[1]

Summary

1) A bedside method is suggested of coupled atrial pacing to slow the rapid ventricular rate during uncontrollable sinus tachycardia.

2) Two indications are defined for paired ventricular stimulation in patients with Stokes-Adams disease: a) persistence of bigeminy with tendency to paroxysmal ventricular tachycardia during single pacing and/or the need for the effect of postextrasystolic potentiation of ventricular contraction; and b) a malfunctioning pacemaker with excessive rate of ventricular stimulation until the latter can be abolished by disabling the "runaway" pacemaker.

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